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ACUTE PULMONARY EMPHYSEMA OBSERVED DURING THE EPIDEMIC OF INFLUENZAL PNEUMONIA AT CAMP HANCOCK, GEORGIA.

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Prior to the epidemic of influenza at Camp Hancock there occurred in one company an outbreak of hemolytic streptococcus infection of peculiar virulence and uniform course. There was reason for thinking that the spread of disease in these cases was by food or milk infection. One man primarily developed this trouble and in a few days six other men were affected simultaneously, all showing aphonia, paresis of the soft palate, inability to swallow on account of choking, substernal pain, but otherwise no other subjective symptoms except cough. The expectoration became more profuse, bronchitis developed and three of the seven patients died. The temperature and pulse in all of these patients was practically normal throughout the course of their illness. Dyspnea developed as bronchitis increased, and it seemed to us that these patients really were drowned by the thick pus which they were unable to expel from the bronchi and trachea. Autopsy in these cases showed a streptococcus infection, with a remarkable affinity for the trachea, and gradually extending down through the bronchi. There was visible peribronchitis and peritracheitis. The larynx and the lungs escaped invasion. This series of cases is mentioned because there were many points of similarity with the bronchitis which accompanied influenzal infection later, and which showed uniformly a bronchitis and peribronchitis very similar to these cases but accompanied by pulmonary emphysema and so-called bronchopneumonia.

During the latter part of September there appeared at the camp an outbreak of disease which did not resemble influenza clinically, but which increased rapidly in frequency until during the last week of that month there were admitted to the hospital about 450 patients with this acute infection of the upper respiratory tract. These showed a mild coryza and conjunctivitis, some pharyngitis, very little bronchitis, frequently mild acute sinus symptoms and some slight cough, mild fever, brief course, and no severe pains. A number of cases of pneumonia developed among the patients. We had, during the epidemic, three wards of these so-called "Camp Hancock" pneumonias which were typical lobar pneumonia clinically. They all showed prune-juice or rusty sputum, high temperature, as a rule, with rapid course and termination by crisis. They all showed typical signs of consolidation in some portions of the lung. In many cases the course was brief after admission to the hospital, and, in fact, some of them seemed to be admitted just prior to crisis. We were able to keep those patients isolated from the bronchitis cases during the epidemic, and while the epidemic bronchitis wards were showing a mortality of nearly 40 per cent., there were only a few deaths among these pneumonia patients (under 5 per cent.) These acute upper respiratory infections, while not considered influenza at that time, appeared to exert a protecting influence when the real influenza appeared, and we were forced to the conclusion that an attenuated influenzal infection must have been responsible for the mild epidemic.

About October 1 there arrived at the camp over 2000 men who had become infected at another camp and had then been subjected to a prolonged railroad journey. The crossing of infections during this long journey produced a profound infection similar in all the patients and presented a very clear-cut clinical picture in this large group of men. We admitted to the hospital in all about 1800 of these cases. They were characterized by:

1. Prostration—incident to the disease and added to by the fatigue, hunger and lack of care unavoidable during the long journey.
2. Cyanosis—particularly of the face and ears and intensely red conjunctivæ and dusky red pharynx.
3. Dyspnea—some degree of dyspnea was invariable; breathing was very shallow.
4. Epistaxis—very frequent, probably in nearly half the cases, and in many instances severe in degree.
5. Bloody sputum.
6. Cough—all had cough and showed signs of bronchitis in varying degrees of severity.
7. Extreme lethargy—a typhoid state, clear sensorium, but apathy and drowsiness; very few were delirious.

The patients suffering from influenza who recovered without marked pulmonary complications were, as a rule, convalescent within a few days and did not experience the nervous or renal complications which have been reported as so frequent in previous epidemics. Many of these men developed their bronchitic emphysema during the first day or two of influenza, others were apparently convalescent from the influenza and seemed ready for discharge to duty when there was a sudden onset of the so-called pneumonia, with characteristic change in the physical findings.

In the so-called pneumonic cases there was evident absent breathing, with impaired resonance at the bases and hyperresonance over the rest of the chest. We were struck with the burly appearance of these patients. This appearance was due to the distention of the neck vessels and erection of the chest, which was dilated to the full inspiratory limit. These patients showed increased temperature, with bloody sputa, and large areas of absent breath-sounds, diminished vocal fremitus, and above these areas crepitant rales in circumscribed zones and diffuse bronchitis throughout the lungs. Forced breathing might bring out rales where sounds had been absent. If breath-sounds reappeared the expiration was markedly prolonged. In no case was the sputum of the rusty or prune-juice type, but was foamy and bloody, and sometimes mixed so completely as to resemble tomato purée. Areas of dulness appeared in the bases, gradually spreading from below upward, and the sputum became frankly purulent or remained bloody, but was not viscid, as in pneumonia.

The temperature was irregular, always elevated but not unduly high; these patients might have sweating throughout the entire course of the disease. The pulse was slow for the temperature, suggesting reflex vagus inhibition from pulmonary distention. There was no arrhythmia and the heart remained apparently good to the end. With the finding of dulness in the bases, which might occur within a few hours of the appearance of the areas of absent breathing, patches of tubular breathing would appear as in bronchopneumonia. These small areas of dulness rapidly coalesced until the whole base of the lung showed signs as in a lobar pneumonia, with marked percussion changes, sometimes flatness, and tubular breathing with egophony and whispered pectoriloquy, as in lobar pneumonia, often without rales.

The rapid massive involvement was accompanied or even preceded by the generalized pulmonary emphysema. Whether this was due to inflammation of the lung parenchyma, causing a loss of tone, or to nervous or circulatory trophic changes, causing loss of tonicity, or to bronchial blockage with valve action production is not clear and will not be discussed here. The sudden occurrence of profound emphysema in these cases as the outstanding feature of the condition was unmistakable. At this stage of consolidation, as at the prior stage of absent breathing, there was evident above the thus affected area a portion of the lung which was apparently compressed and showed diminished or absent breathing, impaired percussion note, and on forced breathing, or after hard coughing, showers of fine crepitant rales as the compressed lung unfolded. We consider this sign of changing areas of compressed lung a fairly constant sign of emphysema. The total lung volume is too great for the chest capacity, and some portion of the lung is usually compressed with other portions fully distended, these areas changing after deep breathing or coughing, with breath-sounds and percussion resonance being reëstablished in the expanding portions.

The areas of consolidation appeared usually posteriorly almost always on one side, followed by the other, spreading and coalescing toward the lower limits of the lung until the base was universally involved, then gradually spreading upward, one side more than the other, but seldom with one side wholly free from involvement. The total consolidation appeared as a gradual filling from below up. Bronchitis persisted throughout, but tubular breathing and pectoriloquy became pure in the solid portion, reaching perfection at the lower levels. Subcrepitant rales were typical in some portions.

The point that struck us here with great force was the intense dyspnea, with little cardiac disturbance, cyanosis, great air hunger, and erection of the chest fixed in a state of hyperinspiration, with only tidal air movement. As the muscles of respiration failed exitus occurred, a respiratory death, in contradistinction to the toxic circulatory or vasomotor death commonly seen in the early days of true pneumonia. The pulse impressed us as being slowed as if under the influence of cardiac vagotonic inhibition, as in high pulmonary pressure. The pulse in desperately ill or moribund individuals was excellent in volume and quality, and relatively slow; arrhythmias were almost never noted. As this distention of the chest developed it could be noted that the whole chest excursion was diminishing as manifested by a general diminution of the lateral excursion of the costal arches until there was an inspiratory retraction, more in the affected side at the onset, then becoming bilateral.

Very soon the accessory muscles of respiration were the only effective ones giving a lift of the upper chest, while over the rest of the thorax was seen only a weak effort of the muscles, giving an undulatory movement of the chest, already so overdistended that any gain in capacity was impossible. Sternal tympany became more marked, assuming a box-like character. Marked stasis of all the veins entering the chest and those drained by the superior vena cava were manifest. Cyanosis became more profound and in the end stage there was an ashen lividity of the whole face and chest.

When in apparent respiratory extremis, frequently a patient would begin to complain of pains, substernal and in the jugular fossæ, and crepitation would be noted in the subcutaneous tissue at the root of the neck, and immediate marked subjective relief was apparent, rapidly followed by a noticeable improvement in the respiratory excursion of the chest, and the most striking decrease in cyanosis and jugular distention. With the relief of cerebral venous stasis a clearing up of the previous accumulative mental hebetude occurred with remarkable suddenness. Cutaneous emphysema soon became diffuse, not infrequently spreading down the trunk to the pubes, first appearing along the sheaths of the cutaneous veins. As the intrathoracic pressure was thus relieved these apparently moribund patients often went on to recovery. We then began measuring the respiratory excursion, the measurements being taken by Lieutenant Beveridge, and his measurements verified these clinical observations, in that there was apparent an increase in the chest circumference during the illness and an abnormally small respiratory excursion, with a striking increase immediately as the cutaneous emphysema gave vent to the air contained in the mediastinum.

The emphysema of the lungs and mediastinum was shown by the marked sternal and vertebral tympany, the disappearance of cardiac dulness and the low stand of the diaphragm, with the pleural sulci distended in full, marked limitation in the motion of the ribs, inspiratory retraction of the costal arches and marked cyanosis. The extent of the emphysema was so great that the supraclavicular and suprasternal fossæ were obliterated and the upper interspaces bulged on expiration and markedly on coughing. It was difficult to outline consolidations in the bases, because the percussion note over the whole chest was so hyperresonant and also because the lung tissue was found to be air-containing.

The course of the disease was entirely different from that of lobar pneumonia. This contrast was particularly striking, as we had at this time three wards of pneumonia patients, all of whom showed typical prune-juice sputa and termination of acute, severe, frequently toxic courses by typical crisis. These cases, which were kept carefully isolated from newcomers, recovered without marked bronchitis and furnished an opportune contrast for comparative observation. We could not fail to be impressed by the presence in one group and the absence in the other of the characteristics noted above: namely, the peculiar type of sputum, cyanosis, dyspnea, respiratory movements and extreme emphysematous fixation of the chest, almost as in an animal in anaphylactic shock. Most of about 1800 patients received after a long railroad journey, with a profound degree of infection and thorough crossing of all the infectious strains, showed some degree of bronchitic emphysema and gave us an unusual opportunity for observing a condition developed to its greatest degree of uniformity and intensity.

The sequence in these cases is usually as follows: At the onset signs of generalized bronchitis and emphysema, universal throughout the lungs and most marked at the bases and toward the roots of the lungs. This is the stage of absent breath-sounds in the area which is about to show signs of consolidation.

Roentgen-ray plates taken at this stage are peculiar in that while the percussion note is resonant or hyperresonant throughout the chest, and the breath-sounds are diminished in intensity and not roughened, the skiagraph shows a diffuse clouding of the whole lung, with the bronchi clearly outlined, broad and dense, giving a streaked appearance, as in a moderate degree of bilateral fibroid phthisis.

The next stage showed continued universal bronchitis, with patches toward the bases showing the auscultatory signs of consolidation, which rapidly coalesced until these areas became diffuse, rapidly advancing upward, always bilateral, though the involvement in one side might be limited in extent. These signs were particularly well-marked in the back. The percussion note was altered by the emphysematous condition of the lung, so that a true flat note as elicited in pneumonic consolidations was not found. A note might sound almost flat in comparison with the hyperresonant or tympanitic note found in other portions of the chest, but careful analysis always has shown that there was a large factor of resonance or high-pitched tympany when this area was considered by itself. It may be noted here that these apparently solid lung portions all were air-containing and floated high in water. This is the stage in which the tympanitic note on the sternum becomes so marked, cyanosis and dyspnea extreme, the chest erected to its greatest capacity as if filled with a lung which is too large for the thoracic cage and the respiratory excursion practically obliterated. Toxic signs were not evident and pulse and temperature relatively low as compared with pneumonia. Delirium was comparatively infrequent and the anxiety and mental distress so marked in pneumonia were strikingly absent.

A number of the patients dying in this stage of early emphysema came to autopsy. The cause of death was evidently the mechanical obstruction of the return flow of the venous blood from the general circulation to the interior of the chest by the thoracic fixation interfering with the mass movement of the blood. Dilatation of the right heart cannot be demonstrated clinically in these cases possibly because of the overlying emphysematous lung and tympanitic sternum, and probably because these hearts are not dilated as in pneumonia and certainly not sufficiently dilated to account for the enormous distention of the superior and inferior vena cava as a prime factor in this stasis. This stasis in the large veins appeared to be due to the obstruction occasioned by increased intrathoracic pressure, and the relief of stasis following immediately upon the escape of air from the mediastinum into the loose areolar tissue surrounding the large veins was one of the most striking features of the whole series of observations and was comparable to the relief afforded by venesection in the distress of acute cardiac dilatation.

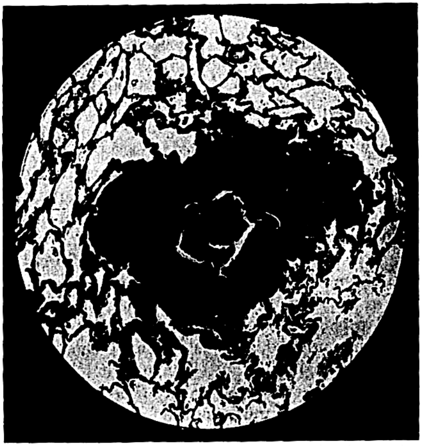

FIG. 1.—Bronchitis, peribronchial infiltration and acute emphysema, with turgescence and infiltration of the alveolar walls.

While we had many autopsies showing this stage of the disease, one of the most graphic demonstrations of the extent of this emphysematous dilatation was afforded by the necropsy finding in the case of patient "G." In this case true lobar pneumonia supervened as a reinfection on an existing bronchitic emphysema and involved the upper and middle lobes on the right, making a perfect cast, measuring 40 cm., on the convex surface, 24 cm. on the concave and

15 cm. at the hilus. The rib markings deeply indented the surface. This cast by preventing any collapse of the lung showed the enormous distention which these lungs can attain.

The lungs of bronchitic emphysema at this stage showed a great similarity in gross pathology as well as in clinical findings. The lungs were gray toward the apex, with a uniform beefy red appearance towārd the base. The surface was glistening, showed the rib markings and emphysematous bullæ, which were plainly visible under the visceral pleura. The emphysema was such that the lungs could not be collapsed by pressure, and while fairly tough at the apex were extremely friable at the bases. Here the tissue, while apparently solid, was always found to be air-containing, floated in water, and although elastic to a light touch, disintegrated under moderate pressure, appearing almost necrotic.

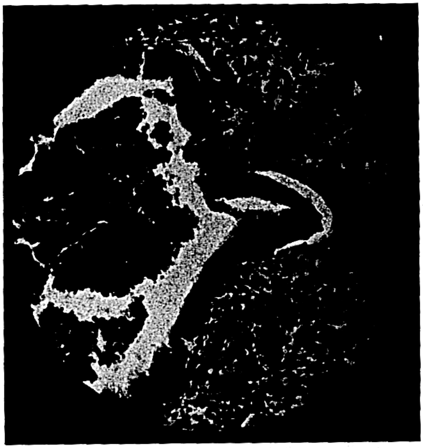

Fig. 2.—Portion of section shown in Fig. 1 under high power, showing changes in the columnar cells lining the bronchus, and the surrounding round-cell infiltration.

The cut surface showed color as on the pleural surface, and from the red portions bloody serum exuded. The surface was glistening and pulled away from the bronchial stumps, which stood out prominently, showing thickened indurated walls and infiltrations surrounding them in cross and longitudinal sections and exuded thick, creamy pus. This poured out in quantity from the large bronchi. All lobes of the lung were studded with nodules from the size of a millet-seed to the size of a bean, which were inflammatory areas surrounding sections of the bronchi and bronchioles. Thick aggregations of these formed irregular nodules, which simulated lobular consolidations; but to the touch these lungs had a feeling which was similar in all of them and quite characteristic. While the nodules varied in number and degree of coalescence they were distinct from lobular pneumonia, and of all pathological lung processes with which we are familiar they resembled only acute disseminated pulmonary tuberculosis. In looking at these lungs it seemed impossible that such lungs could clear up without multiple abscess formation or extensive necrosis. The lung parenchyma in the bases between these inflammatory nodules was mushy and friable. It seemed as though the blood supply and innervation must have been interfered with by the peribronchial infiltrations. The hard nodules were lacking in those cases in which death occurred in the first days of the disease, but the same necrotic character of the lung parenchyma was present and the thickening of the bronchi evident. Pleural involvement was practically absent and empyema not found.

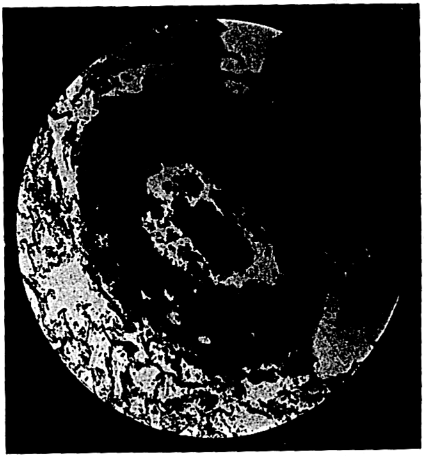

FIG. 3.—Coalescence of peribronchial infiltrations in the evolution of consolidations.

When convalescence was prompt and large abscesses or pleural fluids did not develop, the patients apparently recovered remarkably quickly and did not appear like men who had been through a course of pneumonia. The toxic depression was lacking and they looked remarkably well, but rales persisted throughout the chest and peribronchial fibrosis was marked. From the roentgen-ray plates one would say that there was present much lung destruction and permanent damage.

Other patients who were slow in convalescing showed an irregular temperature curve with febrile exacerbations, continued generalized bronchitis and purulent expectoration. The peribronchial thickenings and infiltrated areas, standing out clearly and separated by emphysematous lung, were so generally distributed and so close together in the roentgen-ray plates of these chests that the descriptive term "snow-storm" was applied to these plates. Multiple small abscesses were always present, thickly strewn throughout the lung, and these patients might die without the development of large areas of necrosis with empyema which frequently terminated the struggle for life.

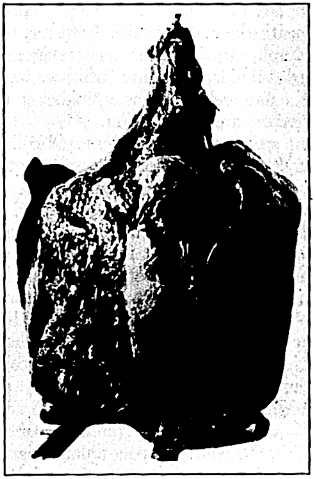

FIG. 4.—Photograph of thoracic contents removed entire, showing large emphysematous bullæ of lung and particularly of mediastinal alveolar tissue. Specimen preserved in Kaiserling's fluid.

We encountered nothing resembling the primary empyema or extensive early pleural involvement accompanying the active pneumonia observed in the epidemics of bronchopneumonia of a year ago. When fluid was found in our cases it occurred late and seemed always due to a direct extension to the visceral pleura from an underlying inflammatory focus. The fluid always occurred late and if explored when first appearing was usually clear straw colored and coagulated promptly on withdrawal. If such a fluid was withdrawn by aspiration almost invariably sticky frictions were audible within a few hours. If reaccumulation of fluid occurred it was, as a rule, distinctly purulent or sometimes fibrinous, making aspiration difficult.

Collections of fluid were in thin layers as if they had accumulated in the pleural cavities under pressure of the emphysematous lungs, which afforded insufficient space for the usual localization in the dependent portions of the chest and forced the fluid along the paths of least resistance, frequently upward and along the anterior mediastinum.

There was dulled tympany corresponding to the fluid, but percussion alone gave little distinction between areas of consolidation and fluid. These thin layers of fluid under high tension apparently served as excellent transmitters or conductors of voice and breath-sounds as well as of tympany to percussion from the compressed emphysematous lung beneath them. They did, however, act as mufflers in diminishing the amplitude of the vibratory waves, and we found that pure high-pitched tubular breathing, whispered pectoriloquy and egophony were the most reliable signs of fluid even though tympany was marked over these areas of effusion.

When the chest space was reduced by rapid collection of fluid in the pleural cavity, the emphysematous upper portions of the lung were pressed against the walls of the upper chest. These lungs (as noted invariably at autopsy) could not be collapsed by pressure, and the resulting signs were very different from those found where a normal or non-emphysematous lung is compressed by a large pleural effusion. We noted in these cases signs practically indistinguishable from those found in large cavities or open pneumothorax. The apices gave a perfect tympany to percussion and cracked-pot sound and a ringing character of the spoken voice, with amphoric transmission of tracheal breath-sounds. These localized fluid collections, rapidly walled off and under pressure, were frequently accompanied by severe diffuse pain in the axillary region of the affected side, with skin hyperesthesia and tenderness in the interspaces. It was noted that in the 6th, 7th, 8th, and 9th spaces, particularly the latter, the pain was constant even in the absence of respiratory motion, but accentuated on deep breathing or coughing.

The fluids varied in their bacteriological flora and in their physical characteristics. They were uniform in their late occurrence, sometimes six weeks from the onset of the bronchitis, from the fact that they were not the result of a diffuse pleuritis but started as exudates from relatively small areas of pleura overlying the inflamed or necrotic lung tissue. There was uniformity in the rapidity of formation of adhesions; indeed, so far as we could judge from physical signs and from autopsy findings the fluid was often found after adhesions had established themselves and started as softening in a mass of plastic exudate. Massive empyema was hardly ever encountered. A number of cases at autopsy showed the lung tissue at the bases so necrotic that we looked carefully in other patients for signs of lung rupture and consequent empyema. This did not occur early, but eventually a number of patients showed rupture of lung abscesses into the pleural cavity. This rupture occurred after adhesions had formed and the empyemata thus developed were strictly localized and in some cases the bronchial drainage was sufficient to clear them up, at least temporarily. These patients with ruptured lung abscess frequently succumbed after a more or less prolonged septic course. The auscultatory signs of these conditions were well-marked and resembled those of large tuberculous cavitations, with whispered pectoriloquy and bubbling rales very close to the ear. The destruction of lung tissue by necrosis and abscess formation in these cases, as shown at autopsy, was enormous, almost total in the bases; usually parts of the upper lobes were also destroyed, the remaining lung tissue being markedly emphysematous.

Regarding treatment it must be said that no routine treatment was shown to be of value after the bronchitic emphysema was established. In view of the apparent immunity enjoyed by individuals who had recently suffered from attacks of probable influenza of a mild form it would seem that a proper vaccine might be of prophylactic value when influenza is epidemic. Creosote in full dosage in the influenza cases may be of service in preventing the development of bronchitis.

When bronchopneumonia with bronchitis and emphysema is established the therapeutic measures which are useful in the management of lobar pneumonia are of no avail. The emergency is due to mechanical rather than toxic causes, and the problems presented are totally different.

SUMMARY. Observation of 1150 soldiers with epidemic influenza pneumonia at Camp Hancock has indicated that the disease as it occurred there differed so essentially in its pathology and course from lobar, broncho- or lobular pneumonia, according to the usual conception of these diseases, that we designated the condition as Acute Bronchitic Emphysema.

Certain conditions were invariably present, including an intense bronchitis and peribronchitis similar to that found in a previous epidemic of pure hemolytic streptococcus infection. There was also present from the first a destructive softening of the lung parenchyma. In addition to this there was always an early and persisting generalized pulmonary emphysema which frequently was the main factor in causing death by interference with the mass movement of the venous blood. These conditions were found in every case examined at autopsy. Except for frequent acute otitis media there were almost no complications or sequelæ outside of the chest.

The pulmonary emphysema, with consequent venous stasis, accounted for the cyanosis, epistaxis and fixation of the chest in the phase of extreme inspiration, with low stand of the diaphragm, which characterized these cases, and also accounted for the paradoxical physical signs in cases in which fluid developed in the chest.

PRIMARY CARCINOMA OF THE THIRD PORTION OF THE DUODENUM.[1]

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Primary carcinoma of the duodenum is a rare disease. Only 7 cases are reported by Schleringer in 42,000 autopsies made in Vienna. Perry and Shaw in studying the Guy's Hospital Reports found only four examples in 18,000 autopsies. Out of 19,500 autopsies done at the London Hospital 18 cases were found.

Carcinoma of the duodenum comprises about 2 per cent. of the cases of primary malignant disease of the intestine. The disease may occur in any part of the duodenum. Of Fenwick's 57 collected cases the first part of the duodenum was involved in 21.5 per cent., the second part in 57 per cent., and the third in 13.5 per cent.

The majority of these tumors belong to the cylinder-celled type, but colloid cancer and spheroidal-celled types occur. Adenocarcinoma usually encircles the gut and causes a constriction of its lumen, while the spheroidal-celled carcinomata either take the form of soft excrescences or of deep ulcers, with elongated edges and fungated bases.

This case was an adenocarcinoma, involving the third portion of the duodenum below the biliary papilla. It encircled the bowel wall and produced almost complete occlusion. It was primary and no other organ was involved. Before proceeding to give the history of the case, I wish to review the symptomatology of the disease. The clinical picture presented depends upon the location of the growth in the duodenum. Carcinoma above the biliary papilla—namely, in the horizontal portion of the duodenum—presents the symptoms of pyloric stenosis, beginning with discomfort and flatulence after meals, with later pyrosis, loss of flesh, anemia and finally vomiting of a persistent type, with or without hematemesis. A palpable mass is usually present. Carcinoma in the vicinity of the biliary papilla—namely, in the second part—is ushered in with a slow developing icterus without pain or vomiting. Chills and fever may be present. The jaundice persists, vomiting develops, the liver becomes enlarged, the gall-bladder may be distended and palpable, there is progressive loss in weight and strength and the patient dies in inanition. A palpable mass may or may not be present.

In carcinoma of the third portion of the duodenum the first symptoms, according to Fenwick, consist of flatulence and discomfort after meals, burning acid eructations, loss of appetite and gradual emaciation. After a lapse of a few months vomiting appears and persists until the end. (In this case the vomiting was intermittent and there were months in which the patient was free from it.) The tongue becomes thickly coated and thirst becomes excessive. (In this case the tongue was clean.) There is marked cachexia and rapid loss in flesh. (In this case no real cachexia and no persistent loss in weight until near the close, but, on the contrary, a gain of eight to ten pounds during one interval of the illness.) The bowels are obstinately constipated. (In my case the bowels could always be moved by a saline.) The stools are seldom quite devoid of bile and often contain altered blood.

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[1] Presented before the Minnesota Pathological Society.

The character of the vomiting is an important feature. It always contains bile, which gives it a bright green color. The filtered vomited material is neutral or alkaline in reaction. (In this case it was acid.) It contains no free hydrochloric acid, but usually lactic acid. (The contents always contained free hydrochloric acid in the case reported and contained no lactic acid.) Will digest fibrin if warmed, owing to the fact that it contains pancreatic juice. (This test was not made in my case.) From time to time attacks of intestinal obstruction intervene, attended by incessant vomiting and obstinate constipation. At these times from 10 to 15 pints of an alkaline bilious fluid may be vomited in the twenty-four hours, notwithstanding the fact that the patient has taken nothing by mouth.

Examination of the abdomen shows a much enlarged stomach, with pronounced succussion as far out as the right mammary line. No tumor, as a rule, can be palpated unless the retroperitoneal glands are involved, when an ill-defined hard mass can be felt to the right of the umbilicus. The introduction of a stomach tube gives evidence of considerable importance. After the stomach has apparently been emptied a fresh gush of fluid gastric contents appears, with cough, or when the body is bent to the left, due to the regurgitation of the duodenal contents. After the stomach has been evacuated a succussion splash may be obtained to the right of the navel, owing to the presence of fluid in the duodenum. Finally, after emptying the stomach at night, a quantity of bilious fluid may be again extracted in the morning before food is taken.

Case History. Mr. G. H., aged seventy-four years, married, children, a man of large affairs. No cancer history in his parents. Had been strong and well as a young man. For the past seven or eight years had had distress in his upper abdomen and back from time to time. He was a very heavy eater, and these spells were attributed to indiscretions in his diet. The distress was never acute, but he would complain of some ill-defined soreness in the upper abdomen. He would often vomit once or twice. The spells never continued for more than a few days, and would then disappear. During the later years of his life he had been subject to attacks of bronchitis in the winter. In some of these his breathing had bothered him, especially at night, and in one or two he had had some cardiac disturbance, with a weak, feeble, rapid pulse. Had had casts and a little albumin in his urine for years. Had taken a moderate amount of stimulants all his life.

In the latter part of November, 1916, following the death of his wife, it was noticed that he seemed pale in color and lacked his usual vigor and energy. No thorough examination was made at the time, as his attending physician attributed the state of health to the trying circumstances surrounding the death of his wife. He was sent to southern California for the winter. While going over the mountains the nurse noticed that he complained of distress in his chest and became pale and weak. After reaching Santiago he was thoroughly examined by an internist and a diagnosis of pernicious anemia was made. No nucleated reds were, however, found in his blood. The blood examination at that time showed 2,500,000 reds and 55 per cent. to 60 per cent. hemoglobin. He had some gastric symptoms in January, 1917, and one or two vomiting spells. In February he had more gastric distress, with vomiting lasting over a month's time. From this he recovered and felt splendidly for about a month, when he again had an attack of persistent vomiting lasting three or four days. He retained nothing upon his stomach during this attack, and the vomited material was dark brown in color. In this attack he vomited large quantities of fluid—in fact, more than he took in. He returned to Minneapolis in May, much improved, and the blood examination, made by his physician, showed 4,000,000 reds and hemoglobin 56 per cent.

I saw him first in June, 1917. He was in one of his vomiting attacks. Had been vomiting two days or so. Was getting up large quantities of dark and greenish-looking fluid, with many black, partially digested blood-clots in it. The fluid vomited was much more than he was ingesting. Would vomit a bowlful at a time. The appearance of the vomited material suggested cancer of the stomach, and this diagnosis had been considered on account of the man's age; but the absence of a palpable mass, the fact that he had maintained his weight, his perfect relief from gastric symptoms between attacks, his high-grade anemia and the negative roentgen-ray examination of the stomach for carcinoma had been considered sufficient evidence for ruling this out.

The physical examination revealed a large-framed, well-nourished man, with iron gray hair. The color of his skin and mucous membranes showed marked anemia, not the typical waxy color of pernicious anemia. He was weak from the vomiting, but had been up and about at home prior to this attack, and felt good. He complained of no numbness of the hands or feet. Nothing of importance was noted outside of his color, a systolic bruit at the apex of the heart, without cardiac enlargement, and a somewhat emphysematous chest with quiet breath-sounds. No enlarged glands or hard nodules in the skin could be felt. A small lipoma, the size of a bean, was present in the abdominal wall of the upper right abdomen. This at first was confusing and was interpreted as intra-abdominal. No mass could be palpated in the abdomen. The rectal examination was negative. Distention of the stomach with gas showed a large stomach, the greater curvature being below the umbilicus. No mass could be palpated after gas distention. No peristaltic waves could be seen sweeping over the stomach. Marked succussion was present over the stomach area. The liver was not enlarged. The spleen not palpable. No masses could be palpated in other parts of the abdomen.

The examination of the vomited material showed a strongly acid reaction; free hydrochloric acid 6, total acidity 20; no lactic acid; strongly positive benzedine; no Oppler-Boas bacilli. The gastric analysis following an Ewald meal showed free hydrochloric acid 59; total acidity 100; no lactic acid; negative benzedine.

The blood examination showed hemoglobin 45 per cent.; reds, 3,900,000; leukocytes, 7000. Differential count: polynuclears, 71 per cent.; large mononuclears, 2 per cent.; small mononuclears, 23 per cent.; transitionals, 2 per cent.; basophiles, 2 per cent.; no myelocytes; no nucleated reds.

The urine was acid; specific gravity, 1018; contained some albumin; no sugar; an occasional pus cell; a few small, narrow hyaline and granular casts. The stools showed a strong benzedine test for blood.

We had then here to deal with an old man, well-nourished, looking very anemic, complaining of ill-defined gastric symptoms, exhibiting at times periodical attacks of vomiting of large quantities of greenish material, with partially digested blood, whose physical examination revealed a high-grade secondary anemia, without assignable cause except the presence of blood in the vomit and in the stools. Roentgenoscopic examination of the stomach and bowel was refused on account of a former unhappy experience when previous roentgen-ray examinations had been made.

The primary examination was made June 23, 1917. The patient was observed closely from that time until his death in coma, about seven months later, and one year and two months subsequent to the time when he first presented symptoms of anemia. I wish to give you as nearly as possible an outline of the clinical course, with the blood, gastric and stool studies during this period.

The note of June 27 read: I saw this patient at his house last night. Was feeling much better, retaining all his food. Says he does much better on a solid than on a liquid diet. Is now eating toast, soft eggs, breakfast foods, etc. I palpated the upper abdomen. Could feel the little-finger-sized nodule to the right of the median line, close to the right costal margin. It is probably in the abdominal wall. It is tender to touch and not very hard. Could feel no enlarged glands. No evidence of metastatic nodules or anything suggestive of malignant disease.

Note of July 7 read: Examined Mr. G. H. today. Patient is getting on very well on his diet of rice, milk toast and cereals. He has not vomited. He is not losing weight and feels good. In the mid-abdomen, about midway between the tip of the ensiform and the umbilicus, I could feel a deep-lying resistance, which I could not outline. This spot is tender to touch on firm pressure. Examination of the stools shows occult blood. Have placed the patient on treatment for duodenal ulcer.

July 16. Examined patient again today. Says he is comfortable, more so than he has been for a long time. Has had no vomiting. Could palpate no mass or tenderness in the abdomen. Is eating only rice and bread and milk. Is fed every two hours. Is gaining in weight. Occult blood is still present in the stools, but the reaction is not as pronounced as before. Blood examination shows hemoglobin, 64 per cent.; red cells, 3,732,000.

August 28. Patient feels much better. Is almost free from abdominal distress. No vomiting. Sleeps well. Still on soft diet. Is gaining in weight. Stools show occult blood. Can palpate no mass. Hemoglobin, 58 per cent.; reds, 3,253,000.

October 1. Patient looks a trifle paler. Up and about each day. Is complaining of a dull pain coming on from time to time in the lower right abdomen. It does not persist and lasts only for a minute or two. He has complained of this distress frequently. No mass could be palpated. The old area of tenderness in the upper mid-abdomen is not present. No enlarged lymph glands in the groins, axillæ or elsewhere can be palpated. Spleen not enlarged. Liver at the costal margin. Nurse states that for about a week stools have been a dark brown. Stool examinations show occult blood. Blood examination shows a sharp drop: hemoglobin, 36 per cent.; reds, 2,840,000; leukocytes, 5600. Differential count: polynuclears, 74.5 per cent.; large mononuclears, 2.5 per cent.; small mononuclears, 20.5 per cent.; transitionals, 2.5 per cent.; eosinophiles, 0; basophiles, 0; myelocytes, 0; no nucleated reds.

October 6. Examined patient today. Having all stools saved. The stools are lighter and have lost the dark brown color of a few days ago. Put an ice-bag again over the abdomen. The patient has evidently had more bleeding, which is now quieting down.

October 10. Stools are still lighter. Have still a greenish color. Dark brown color has disappeared. Patient complains of ill-defined distress in the upper abdomen. Calls it indigestion. Says it shifts from one point to another. Says it comes on after eating. Belches gas and it disappears.

October 16. Patient is not doing so well. Seems more prostrated and weak. Color is paler. Stools are much lighter, a pale green, but still contain blood by the benzedine test. Blood examination shows hemoglobin, 23 per cent.; reds, 3,184,000.

October 29. Patient is again better. His color is not so pale. The pain in the mid-back, which he has had for a number of days, has disappeared. Palpated the abdomen but could feel no mass. Stools still contain blood. Blood examination shows hemoglobin, 37 per cent.; reds, 3,232,000.

November 18. Examined Mr. G. H. today. Is doing fairly well. His color is better. Has gained some in weight. Complains very little of distress in the upper abdomen. Is eating a fairly general diet without distress. Stools are a typical milk stool, but show occult blood. Went over the abdomen carefully, but could palpate no mass.

December 16. Saw this patient today. Is doing splendidly. Color is improving. Is eating a fairly general diet, including fish and bacon and scraped beef, without distress. Blood examination is better: hemoglobin, 42 per cent.; reds, 4,016,000.

January 6, 1918. Patient is having distress in the upper abdomen, with belching of gas. Thinks it is due to sweetbreads which he had eaten. Has been eating all kinds of foods without distress up to this time. Stools are again a dark brown color and give a stronger benzedine test.

January 8. Patient was seized with a severe attack of vomiting in the night. Vomited, two or three times, a large amount, 500 to 1000 c.c. The material was a typical coffee-ground vomit. In spite of the fact that he took little fluids large quantities of dark material continued to come up. There was no tenderness in the abdomen. A marked succussion sound was present over the stomach area. The vomited material was acid, contained free hydrochloric acid, 12; total acidity, 34; no lactic acid; was strongly positive for blood by benzedine.

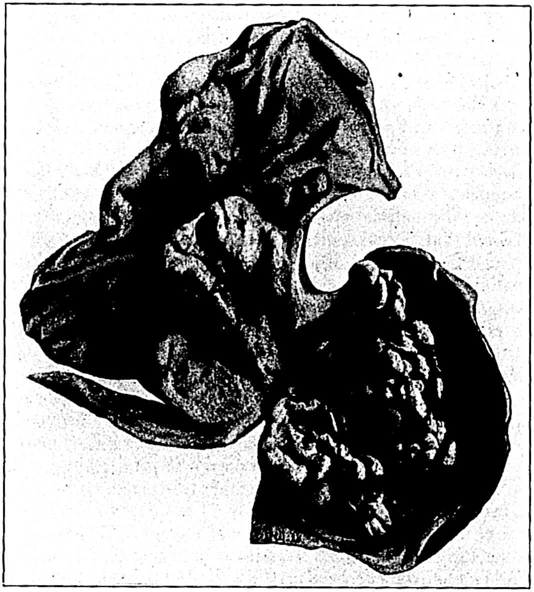
January 11. Patient today vomited a large amount of dark, grumous, black material, depositing a heavy sediment of small dark blood-clots on standing. Some of the last of the vomited material coming up contained bright blood. The vomited material was acid; contained free hydrochloric acid, 4; total acidity, 36; no lactic acid. Strongly positive benzedine test.

January 13. Patient is doing badly. Continues to vomit about 1000 c.c. of dark coffee-ground material four or five times a day. The vomiting attacks come in spells. Is taking plenty of fluids, and when the stomach becomes filled begins to vomit. Vomited material has no fecal odor. The vomiting, however, is of the obstructive type, as the stomach can be seen to slowly fill up with fluids and then empty itself, only to be filled again. Patient is rational, his pulse is good and the supply of fluid by rectum relieves his thirst.

January 17. Patient is losing ground. Vomiting still continues, all of a coffee-ground character, and in large amounts. He is becoming weak and prostrated. Anemia is marked. Patient is perfectly rational and is in no severe pain. The vomiting gives him relief.

January 19. Patient died today in coma. Vomited coffee-ground material only a few hours before his death.

With the completed clinical picture of the disease before us, one of two interpretations seems justified in a man aged seventy-four years, showing a high-grade anemia, with blood persistently in the stools, a hyperacid gastric content, no palpable mass, and dying with the symptoms of obstructive vomiting—namely, either a carcinoma high in the intestinal tract below the stomach or a duodenal ulcer with a carcinoma superimposed, causing obstruction by occlusion of the bowel. The diagnosis of the case prior to autopsy was a duodenal ulcer, with carcinoma superimposed, producing a slow-developing occlusion of the intestine not far from the pyloric opening.



Primary carcinoma of the duodenum. *A*, carcinoma; *B*, duodenal walls.

POSTMORTEM REPORT. (By E. T. Bell, M.D., Professor of Pathology, University of Minnesota.) Autopsy limited to an abdominal incision. No evidence of disease in the peritoneal cavity. At the lower curve of the duodenum and extending over into the third portion a resistant mass was palpated. The stomach and upper part of the duodenum showed a moderate degree of distention.

Upon opening the duodenum a tumor mass was found situated mainly in the third portion and in the lower part of the descending portion. The lumen was almost completely occluded by a cauliflower-like growth growing from the median and posterior wall. This growth extends from a point 2 cm. below the papilla of Vater to a point 9 cm. below the same structure, the length of the tumor in the long axis of the duodenum being therefore 7 cm. The greatest height of the growth projecting into the lumen of the duodenum is 3 cm. At a point 4.5 cm. below the papilla of Vater the lumen of the duodenum is extremely narrowed, being almost completely obstructed. The point of attachment of the cauliflower-like growth to the inner posterior wall of the duodenum measures 4 cm. There is no involvement of the head of the pancreas, which lies in contact with the portion of the duodenum from which the tumor is growing. There is no enlargement of the lymph nodes in the abdominal cavity at any point.

Microscopic examination of the tumor shows a typical adenocarcinoma of the papillary type. Infiltration extends deep into the muscular layer of the duodenum on its inner aspect.

The kidney shows numerous coarse pits over the surfaces, particularly marked along the convex borders. There is, however, only a moderate reduction in the amount of renal cortex. Microscopic examination shows the lesion to be of the coarse arteriosclerotic type. There is no anatomical evidence suggestive of renal insufficiency.

No gross or microscopic lesions in the adrenals, liver, pancreas or lymph nodes around the celiac axis.

REPORT ON THE EXAMINATION AND TREATMENT OF SOLDIERS INFECTED WITH HOOKWORM AT CAMP BEAUREGARD, LA., MARCH 1 TO SEPTEMBER 1, 1918.

By Donald J. Frick, Major, M. C., U. S. A.,

This report is made for the purpose of showing the wide distribution of hookworm among Southern troops, the necessity of early examination and intensive and repeated treatment.

On account of a number of unavoidable circumstances, the examination for hookworm was not begun until the end of February, 1918. As the work was new to nearly all concerned, it was slow and not systematic. As a result, rather a large number escaped having their stools examined. After the examination was made and the list of positive cases fully determined, it was most difficult to get the men sent to the Base Hospital for treatment. The hospital had plans made to treat 100 cases every forty-eight hours, but was